10/705,448
LVCook 9/15/06
updated Search

d his

(FILE 'HOME' ENTERED AT 11:15:54 ON 15 SEP 2006)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, JAPIO' ENTERED AT 11:16:14 ON 15 SEP 2006

L1 18411 S ATHEROSCLER? AND PLAQUES
L2 119 S L1 AND (LIPID POOL)
L3 62 DUPLICATE REMOVE L2 (57 DUPLICATES REMOVED)
L4 21 S L3 AND PD<2000
L5 2 S L4 AND ANTIBOD?

d his

(FILE 'HOME' ENTERED AT 11:15:54 ON 15 SEP 2006)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, JAPIO' ENTERED AT 11:16:14 ON 15 SEP 2006

L1 [illegible] S ATHEROSCLER? AND PLAQUES
L2 [illegible] S L1 AND (LIPID POOL)
L3 62 DUPLICATE REMOVE L2 (57 DUPLICATES REMOVED)
L4 [illegible] S L3 AND PD<2000
L5 3 S L4 AND ANTIBOD?

ANSWER 2 OF 3 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

AN 90308825 EMBASE

DN 1990308825

TI Atherosclerotic plaque rupture and thrombosis. Evolving concepts.

AU Fust r V.; Stein B.; Ambrose J.A.; Badimon L.; Badimon J.J.; Chesebro J.H.

CS Division of Cardiology, Mount Sinai Medical Center, One Gustave L. Levy Place, New York, NY 10029, United States

SO Circulation, (1990) Vol. 82, No. 3 SUPPL., pp. II-47-II-59. .
ISSN: 0009-7322 CODEN: CIRCAZ

CY United States

DT Journal; Conference Article

FS 005 General Pathology and Pathological Anatomy
006 Internal Medicine
014 Radiology
018 Cardiovascular Diseases and Cardiovascular Surgery

LA English

SL English

ED Entered STN: 13 Dec 1991
Last Updated on STN: 13 Dec 1991

AB Rupture of an atherosclerotic plaque associated with partial or complete thrombotic vessel occlusion is fundamental to the development of ischemic coronary syndromes. Plaques that produce only mild-to-moderate angiographic luminal stenosis are frequently those that undergo abrupt disruption, leading to unstable angina or acute myocardial infarction. Plaques with increased lipid content appear more prone to rupture, particularly when the lipid pool is localized eccentrically within the intima. Macrophages appear to play an important role in atherogenesis, perhaps by participating in the uptake and metabolism of lipoproteins, secretion of growth factors, and production of enzymes and toxic metabolites that may facilitate plaque rupture. In addition, the particular composition or configuration of a plaque and the hemodynamic forces to which it is exposed may determine its susceptibility to disruption. Exposure of collagen, lipids, and smooth muscle cells after plaque rupture leads to the activation of platelets and the coagulation cascade system. The resulting thrombus may lead to marked reduction in myocardial perfusion and the development of an unstable coronary syndrome, or it may become organized and incorporated into the diseased vessel, thus contributing to the progression of atherosclerosis. In unstable angina, plaque disruption leads to thrombosis, which is usually labile and results in only a transient reduction in myocardial perfusion. Release of vasoactive substances, arterial spasm, or increases in myocardial oxygen demand may contribute to ischemia. In acute myocardial infarction, plaque disruption results in a more persistent thrombotic vessel occlusion; the extent of necrosis depends on the size of the artery, the duration of occlusion, the presence of collateral flow, and the integrity of the fibrinolytic system. Thrombi that undergo lysis expose a highly thrombogenic surface to the circulating blood, which has the capacity of activating platelets and the coagulation cascade system and may lead to thrombotic reocclusion. Measurements aimed at reversing the process of atherosclerosis via cholesterol reduction and enhanced high density lipoprotein activity are encouraging. Active research is being focused on the development of new antithrombotic tools, such as inhibitors of thrombin, thromboxane, and serotonin receptor antagonists, and monoclonal antibodies aimed at blocking platelet membrane receptors or adhesive proteins. These compounds may prove useful when immediate and potent inhibition of the hemostatic system is desired. Intensive research is still needed in the areas of pathogenesis and therapeutic intervention in atherosclerosis.

CT Medical Descriptors:
* acute heart infarction: DI, diagnosis
*acute heart infarction: ET, etiology
*angiography

*unstable angina pectoris: DI, diagnosis
*unstable angina pectoris: ET, etiology
ultrastructure
human
conference paper
priority journal